High-Dose Vitamin C | Anti-Cancer Agent

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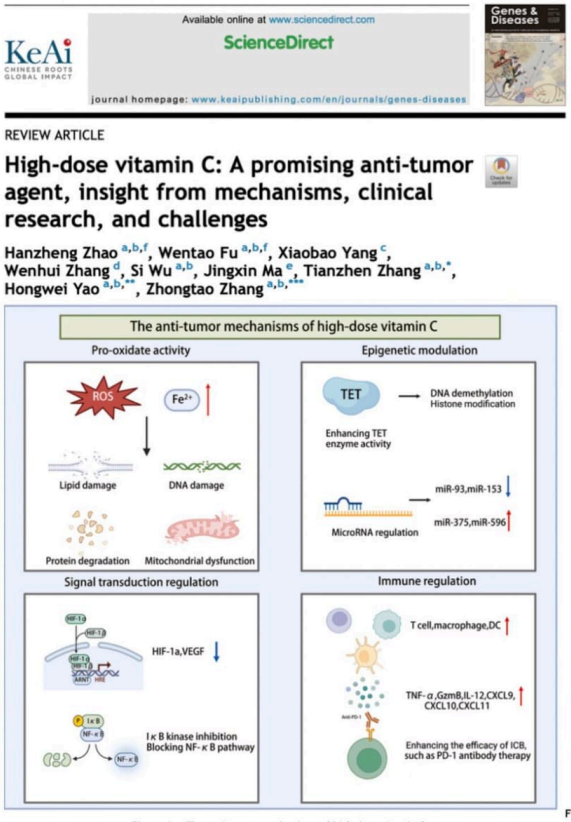

Figure 1 The anti-tumor mechanisms of high-dose vitamin C.

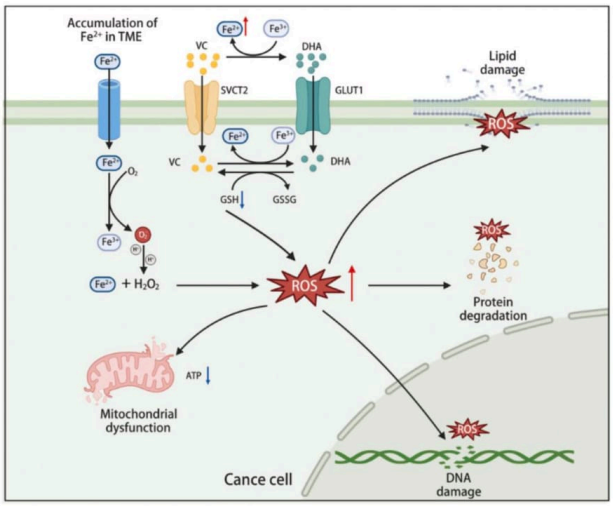

Figure 2 Elevated intracellular oxidative stress induced by high-dose vitamin C disrupts tumor cell redox balance and kills tumor cells.

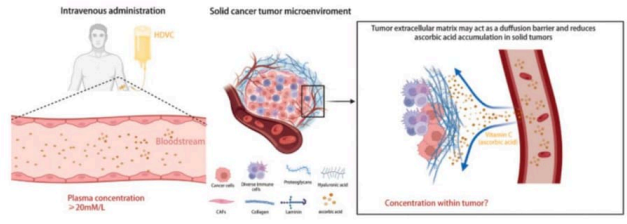

Figure 3 Potential mechanisms underlying the difference between intra-tumoral concentration and plasma concentration of vitamin C.

> **Pharmacologic intravenous vitamin C is a multi-mechanistic, tumor-selective anti-cancer therapy that has been under-dosed, under-studied, and consistently underestimated.**

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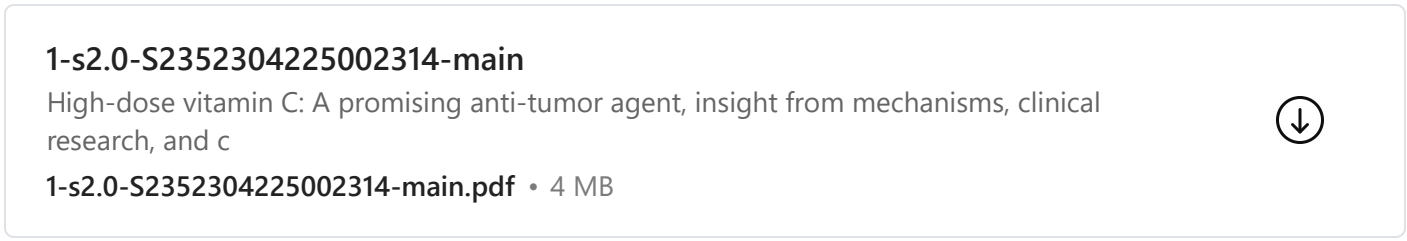
A major new review paper titled, **High-dose vitamin C: A promising anti-tumor agent, insight from mechanisms, clinical research, and challenges**, analyzed 150+ studies and found that when vitamin C reaches true pharmacologic levels (20–30 mM), it behaves like a targeted, tumor-selective therapy — something past trials missed by under-dosing. The evidence base spans decades of laboratory, animal, and early-phase clinical research.



The authors outline four major anti-cancer mechanisms of high-dose vitamin C — pro-oxidative tumor cytotoxicity, epigenetic reprogramming, suppression of oncogenic signaling pathways, and powerful immune activation.

For a therapy this safe, inexpensive, and mechanistically potent, the findings are striking. Here's what they found:

Vitamin C Becomes a Cancer-killer at High Plasma Concentrations

When plasma levels reach the **20–30 mM range**, vitamin C switches from antioxidant to **pro-oxidant**, generating hydrogen peroxide and hydroxyl radicals *inside tumors* — while sparing normal tissues.

Tumors are uniquely vulnerable because they accumulate:

- excess labile iron
- high GLUT1 expression (massive vitamin C uptake)
- weakened redox defenses

This combination creates a selective chemical trap that cancer cells cannot escape.

KRAS- and BRAF-mutant Cancers Are Especially Sensitive

The review highlights a major vulnerability: KRAS- and BRAF-driven colorectal, pancreatic, and lung cancers are selectively disrupted — even destroyed — by pharmacologic vitamin C.

These mutations cause:

- extreme GLUT1 overexpression
- glycolytic addiction (the “Warburg effect”)
- rapid NADPH and glutathione depletion

This makes the cancer cells especially sensitive to vitamin C–induced metabolic collapse.

A Phase III clinical trial even reported **significantly improved survival** in KRAS-mutant colorectal cancer patients when high-dose intravenous vitamin C was added to standard therapy.

Vitamin C Suppresses HIF-1α — The Master Switch of Tumor Survival

Vitamin C is a required cofactor for the enzymes that degrade **HIF-1α**, a central regulator of tumor aggressiveness. At pharmacologic doses, high-dose intravenous vitamin C shuts down:

- angiogenesis
- metastatic signaling
- hypoxia tolerance
- GLUT1 upregulation

Very few agents directly dismantle this survival pathway.

Vitamin C Reprograms Cancer Epigenetics

Pharmacologic vitamin C reactivates **TET enzymes**, reversing abnormal DNA hypermethylation and restoring tumor-suppressor gene expression.

Multiple studies show induced differentiation and suppressed proliferation following high-dose intravenous vitamin C exposure.

Vitamin C Enhances Anti-tumor Immunity

High-dose intravenous vitamin C also strengthens the immune system's ability to attack cancer. It has been shown to:

- Increase $CD4^+$ and $CD8^+$ T-cell infiltration into tumors
- Boost granzyme B and IL-12, enhancing cytotoxic activity
- Upregulate CXCL9/10/11, drawing more tumor-infiltrating lymphocytes
- Synergize with PD-1 and CTLA-4 checkpoint inhibitors
- Enhance T-cell function and proliferation
- Improve natural killer (NK) cell cytotoxicity
- Activate dendritic cells, strengthening antigen presentation

Together, these actions amplify immune-mediated tumor destruction.

Early Clinical Trials Show Meaningful Survival Improvements

The review summarizes multiple Phase I/II trials where high-dose intravenous vitamin C strengthened standard therapy:

- **Pancreatic cancer:** major tumor shrinkage in 8/9 patients

- **Glioblastoma:** median overall survival increased from 14.6 → 19.6 months
- **NSCLC:** response rates roughly doubled
- **Ovarian cancer:** reduced chemotherapy toxicity + longer progression-free survival

Across all studies, safety and tolerability were excellent.

Dosing

The review identifies the dosing regimen required to achieve tumor-selective, cytotoxic plasma levels:

75–100 grams IV per infusion, or >1.0 g/kg IV per infusion

Given 2–3 times per week for 6–8 cycles.

This reliably produces **≥20 mM plasma concentrations** — the range associated with selective cancer cell killing — while remaining well-tolerated.

The authors also emphasize that most patients in clinical trials never reached the maximum tolerated dose, suggesting the therapeutic ceiling is likely far higher than what past studies explored.

This paper makes something very clear: **Pharmacologic intravenous vitamin C is a multi-mechanistic, tumor-selective anti-cancer therapy that has been under-dosed, under-studied, and consistently underestimated.**

Given its safety profile, low cost, and robust mechanistic data, high-dose vitamin C urgently warrants modern Phase III trials with appropriate pharmacologic dosing regimens.

Study Source:
https://www.sciencedirect.com/science/article/pii/S2352304225002314?via%3Dihub

Original Article: https://www.globalresearch.ca/high-dose-vitamin-c-promising-anti-cancer-agent/5907315

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